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SLEEP

Kevin Brewer

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Orsett Psychological Services
PO Box 179
Grays
Essex
RM16 3EW

orsettpsychologicalservices@phonecoop.coop

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Kevin Brewer BSocSc, MSc

An independent academic psychologist, based in England, who has written extensively on different areas of psychology with an emphasis on the critical stance towards traditional ideas.

A complete listing of his writings at http://kmbpsychology.jottit.com.

PAST ISSUES

CONTENTS

Introduction

Siegel (2005) defined sleep as "a state of immobility with greatly reduced responsiveness, which can be distinguished from coma or anaesthesia by its rapid reversibility" (p1264).

Hobson (2005) stated the three fundamental questions about sleep for researchers:

i) What is sleep?
ii) What are the mechanisms of sleep?
iii) What are the functions of sleep?

Until just under half a century ago, it was believed that brain activity was greatly reduced or even absent during sleep. Literally, the person became unconscious during sleep, and nothing happened of interest to psychologists. It was almost as if consciousness ceased at the beginning of sleep and only returned at waking.
But key discoveries challenged this belief:

a) The fact that sleep involves stages and cycles including the distinction between rapid eye movement (REM) and non-REM periods (Aserinsky and Kleitman 1953);

b) The brain is very active during REM sleep, and this is the period associated with dreaming (Dement and Kleitman 1957).

The brain is far from inactive during sleep; for example, the cerebral blood flow is only reduced by 20% during sleep, and as many neurons increase activity as decrease their activity (Hobson 2005).

The study of sleep has been aided by developments in medical technology; initially electroencephalograms (EEG), and subsequently brain or neuroimaging. What these technologies had shown is that there are different patterns of the brain activity. For example, EEG readings during slow wave sleep (SWS) (NREM) produces high-voltage slow waves, and the brain as a whole appears to be inactive. But the brain is still 80% active with intracellular communication in the cortex and thalamus (Steriade et al 1999).

On the other hand, much sleep research is dependent upon subjective reports by sleepers on waking. Memory is affected by sleep, and so reports of mental activity during sleep must be treated with some caution.

Hobson (2005) felt that recent research suggested two key aspects of sleep:

i) Sleep is an active process, "not simply the passive result of diminished waking";

ii) Sleep is not a cessation of neuronal activity, but the reorganisation of it.

All animals studied to date have been found to sleep in some way (Lima et al 2005). EEG readings of mammals and birds show periods of quiet sleep (NREM) and active sleep (REM).

Humans show four stages of NREM sleep, of which the deepest stages (3 and 4) are known as slow-wave sleep (SWS). In rats, for example, two stages of NREM sleep have bee described (Neckelmann and Ursin 1993). All NREM sleep in non-human mammals is usually called SWS (Lima et al 2005).

Birds show changes in the intensity of SWS rather than stages (eg: blackbirds; Szymczak et al 1996).

Humans show two states of REM sleep (tonic and phasic - the latter associated with eye movements), while birds and mammals show only one type of REM sleep (Lima et al 2005).

EEG readings of reptiles show changes in types of sleep, but not necessarily the same as the NREM and REM distinction (Rattenborg and Amlaner 2002). The same is true for sleep in those few amphibians, fish, and invertebrates studied (Lima et al 2005).

Hobson et al (2000) proposed the AIM model to distinguish the three conscious states of REM sleep, NREM sleep, and waking. The states vary on three criteria:

- Activation (A) - information processing capacity;
- Information flow (I) - "the degree to which information processed comes from the outside world and is or is not reflected in behaviour";
- Mode of information processing (M) - the way in which information is processed.

Table 1 shows the combination of the three criteria in different states.

	ACTIVATION (how much)	INFORMATION FLOW (what information)	MODE OF INFORMATION (how processed)
WAKING	high	high	high
DROWSINESS/ QUIET WAKING	intermediate	high	intermediate
NREM SLEEP	intermediate	intermediate	intermediate
REM SLEEP	high	low	low

Table 1 - AIM model and different conscious states.

Measuring Sleepiness

"Sleepiness" is a term with no clear consensus of definition (Shen et al 2006). Traditionally it is measured by subjective and objective methods.

SUBJECTIVE METHODS

These tend to be questionnaires that are self or other administered. The most commonly used in research and clinically is the Stanford Sleepiness Scale (SSS) (Hoddes et al 1973). It contains seven statements for the individual to rate themselves every fifteen minutes: from "(1) feel active and vital; alert; wide awake" to "(7) almost in reverie; sleep onset soon; lost struggle to remain awake". It is an ordinal scale.

The Visual Analogue Scale (VAS) (Monk 1987) asks individuals to rate themselves on a 100mm line with no words other than "alert" at one end, and "drowsy" at the other. Both these measures rate sleepiness at that moment.

The Epworth Sleepiness Scale (ESS) (Johns 1991) attempts to measure inappropriate sleep onset and daytime sleepiness. Individuals rate 0-3 (never - high chance of dozing) for eight situations; eg: "watching TV" or "in a car, while stopped for a few minutes in the traffic". A score of over ten out of twenty-four is seen as "pathological sleepiness".

OBJECTIVE METHODS

These methods are attempting to obtain objective scores of sleep propensity (the likelihood of falling asleep).

i) Multiple Sleep Latency Test (MSLT) (Thorpy 1992).

This is seen as the most objective method. Individuals are placed in a quiet, dark room for 20-30 minutes at two-hour intervals over a 24-hour period. The participant is attached to an EEG machine. Sleep onset within five minutes during the day is defined as "pathological sleepiness".

ii) Maintenance of Wakefulness Test (MWT)(Mitler et al 1982).

Participants are asked at two-hour intervals to sit

upright in bed in a quiet, dark room, and to stay awake. It is very similar to the MLST, but the emphasis is upon staying awake. Inability to stay awake for longer than thirteen minutes is classed as "severe impairment of alertness".

It is interesting that instructions play a key role in these "objective" tests. In the MLST, the average time awake was around twelve minutes in one study, but for the MWT over twenty-five minutes (Mitler et al 2000).

iii) Other measures include pupillometry (measuring the pupil diameter as an individual focuses on a red spot in a darkened room for fifteen minutes); evoked potentials; cognitive and psychomotor function tests (Shen et al 2006).

Table 2 lists the main problems with these methods.

METHOD	PROBLEMS
Subjective eg: Stanford Sleepiness Scale	- "self-reporting of symptoms, leaving it open to misinterpretation, unintended bias and outright falsification" (Shen et al 2006 p65) - Problems of reliability and validity
Objective eg: Multiple Sleep Latency Test	- What is actually measured by MSLT - Artificial situations - Different instructions producing different results between MSLT and MWT - Cut-off points for "pathological sleepiness" are open to debate - Validity of pupillometry as measure of sleepiness - Cognitive and psychomotor function tests measure ability to do tasks not necessarily sleepiness

Table 2 - Some problems with the methods for measuring sleepiness.

Function of Sleep

Siegel (2005) stated that:

> Sleep probably has multiple functions for the brain and body. An important task will be the identification of which of the hypothesized functions may only be achieved during sleep, and which may be executed during both waking and sleep, with sleep being a more efficient time for their accomplishment (p1270).

Horne (2001) felt that the function depended on "evolution, body size, need to conserve energy, cerebral development, and amount of relaxed wakefulness" (pp302-303).

Hobson (2005) noted that all mammals studied show cycles of REM and NREM sleep, "which suggests not only a shared mechanism across species but a universal functional significance..".

Generally it is felt that for humans, NREM sleep is for energy conservation (for the body) and REM sleep is for the brain. However, Siegel (2005) argued that sleep has different functions for different species.

The "argument that sleep serves a vital function is compelling" (Siegel 2005). This can be seen in sleep deprivation studies: for example, rats and flies die quicker from sleep deprivation than from food deprivation (Rechtschaffen 1998). But this is not true for all species, and such results may depend upon the methodology of sleep deprivation used (Siegel 2005).

SLEEP IN MAMMALS

Risk of Predators

Sleep varies between mammals both in terms of the amount, and depth of sleep, and whether some functions of sleep can be achieved during wakefulness (eg: energy homeostasis and conservation).

Sleep as a means to keep the animal safe from predators is one possibility. This idea of the "immobilization hypothesis" was proposed by Meddis (1975; 1977). However, this assumed that sleep had no restorative function, and merely passed the dark hours, particularly during winter.

But this theory cannot explain sleep rebound after a period of sleep deprivation, and the fact that quiet wakefulness could be safer than sleep (Rechtschaffen

1998). Furthermore, if sleep has no restorative function, why are there different types and stages? (Lima et al 2005).

Sleep can change in a number of ways with the risk of predators:

i) More frequent arousals from sleep - Lendrem (1984) increased the risk to doves of a predator, the ferret. The research found that the birds awoke more often to scan the environment, and spent less overall time sleeping. Their normal sleeping behaviour only occurred when the birds were in a group.

ii) Unihemispheric sleep (sleeping on one side of the brain only) - Mallard ducks have the option of unihemispheric sleep in risky situations. Rattenborg et al (1999a; 1999b) increased the apparent risk of predation in a lab experiment where EEG readings could be made. When the fear of a predator increased, there was less overall sleep time, and more unihemispheric sleep (from 10 to 20% of the total sleep time).

iii) Polyphasic sleep (sleeping at different periods during 24 hours rather than in one block - monophasic) or sleeping at different times - Free-living rats changed from sleeping in the day to sleeping at night to avoid red fox predators (Fenn and MacDonald 1995).

iv) Where to sleep - eg: frequently shifting sleeping sites or choosing sites hard for predators to approach.

For Lima et al (2005), "blackout" sleep is a good way to immobilise animals if there is also a restorative function to sleep.

Other Considerations

Sleep has to be a trade-off between staying safe from predators and the need for food. Allison and Cicchetti (1976) compared the sleep patterns of a number of mammals, and produced the following rule: large carnivores slept long and deep, while small herbivores little and shallow (with frequent wakings).

The key being the need for vigilance and the risk of predators as well as the time taken to find food. A lion, for example, is free from predation, and can sleep for long periods between feedings to conserve energy, while a rabbit is vulnerable to many predators and needs a lot of time to forage. For them, sleep is minimal.

Table 3 shows some examples of different animals and sleep time.

ANIMAL	APPROX HOURS OF SLEEP PER DAY
Carnivores: less risk of predation	
Tiger	16
Lion	15
Cheetah	14
Herbivores: high risk of predation and/or foraging takes time	
Elephants	3-5
Sheep	4
Rabbit	8
Omnivores	
Humans	8
Chimpanzees	10
Rats	12-14

Table 3 - Examples of sleep time for different species.

With herbivores, sleep time is inversely related to body mass. For example, elephants have short periods of sleep not because of fear of predators, but, because of their size, finding food takes a long time. While a Mongolian gerbil or a vole sleeps for over twice as long (being small animals).

Siegel (2005) noted caution about the sleep times in different species as they are often based on controlled observation of a limited number of animals at zoos.

To sum up, for terrestrial mammals, length of sleep will be affected by type of diet (carnivore or herbivore), body mass, and brain size (ie: energy required), and risk of predators. Also large herbivores have evolved less sleep than small ones because the former are more vulnerable to predators - "tailoring-of-sleep" hypothesis (Lima et al 2005).

One set of terrestrial mammals are monotremes (ie: egg-laying like the platypus), and they seem to have a different form of REM sleep that only takes place in the brainstem (Siegel et al 1999). Usually in other mammals, REM sleep produces EEG readings throughout the whole brain. Monotreme behaviour is seen in evolutionary terms as closer to reptiles, and this may help in understanding the evolution of sleep.

Marine Mammals

Sleep in marine mammals is harder to study, but

those studied do appear to have different patterns of sleep. For example, cetaceans (whales and dolphins) shows slow-wave (NREM) sleep EEG readings in one hemisphere of the brain at a time (unihemispheric) (Mukhametov et al 1977).

In other words, they are sleeping in half of the brain at a time, and this is supported by the observation that one eye is closed and one open. This has also been found for SWS in some birds.

Also REM sleep has not been found in cetaceans in any studies (Siegel 2005). One important point is that these animals are never immobile, it appears, from birth to death.

Siegel (2005) raised the note of doubt that these animals may not be sleeping. He pointed out that certain drugs produce slow-wave EEG readings in individuals clearly awake. On the other hand, cetaceans may be sleeping more than observed because non-slow-wave EEG readings have been found in NREM sleep in rats (Bergmann et al 1987). This raises the problem in any sleep research of trying to correlate physiological measurements with behaviour in a definitive way, particularly among different species.

This problem makes it difficult to study sleep deprivation in such animals. Oleksenko et al (1992) attempted to prevent sleep in captive bottlenose dolphins by disturbing them whenever slow-wave EEG readings appeared. Looking for evidence of "sleep rebound" (sleeping longer to make up for lost sleep), the researchers found no consistent patterns.

Other differences between cetaceans and terrestrial mammals in sleep have been found. Most mammals sleep a large amount after birth, and this quantity is reduced with age. Not so with killer whales or dolphins where newborns showed little sleep behaviour (Lyamin et al 2005). Also usually mothers sleep longer after birth (post-partum), but this is not seen. Suppression of sleep is evident in birds during migration season without ill effects or rebound (Siegel 2005).

Another marine mammal shows different patterns of sleep still - namely, fur seals. They show a similar pattern of unihemipsheric slow-wave sleep (and a small amount of REM sleep) when swimming, but when on land, their sleep patterns revert to that similar to land mammals (Lyamin et al 1996).

To sum up, sleep in marine mammals has evolved in a different way to that of terrestrial mammals, and is a product of an environment where the animals cannot remain immobile for long periods.

FUNCTION OF NREM SLEEP

Benefits to the cortex

Horne (1993) compared regional cerebral metabolism (rCMR) in the prefrontal cortex (PFC). in the PET scanner during different states of sleep and wakefulness. The PFC is the area of the brain lying in front of the primary and secondary motor cortex, and includes the dorsolateral and orbital areas, frontal eye fields, and Broca's area. Regional cerebral metabolism is used as a measure of brain activity. It was highest during relaxed alert wakefulness, and lowest during stages 3 and 4 NREM sleep (slow- wave sleep). This could mean that SWS aids cortical recovery.

Physiological measures of the brain show that it behaves differently during NREM sleep (eg: EEG readings). So it should be expected that both total sleep time and NREM/REM division will be related to size of cortex.

But this is not the case. The elephant with a large cortex has little sleep compared to the opposite of the rat (Siegel 2005). This is in relation to recuperation of the whole cortex (or brain) during sleep. Recent research suggested the local recuperation of specific areas of the brain based on prior learning during waking (eg: Huber et al 2004).

Vyazovskiy et al (2004) stimulated the whiskers on one side of the face of mice during sleep deprivation. Then during sleep, areas of the brain related to the whiskers stimulated behaved differently to the other parts of the brain.

Other studies have shown biological processes at work during NREM sleep like protein synthesis (Nakanishi et al 1997), and the generation of new cells (eg: Guzman-Marin et al 2003 in the rat hippocampus).

Regulation of the body

The results of long-term sleep deprivation of animals eventually leads to death. But before that occurs, the animal experiences body temperature changes and skin lesions. Both are signs of the regulation of the body being out of control. These effects are the same as hypothalamus damage, and the consequent abnormal functioning of the endocrine and immune systems (Everson and Crowley 2004).

Horne (2001) argued, that for humans, there is little effect on the body of sleep deprivation. Most of the detriment is to the brain.

Other research suggested that sleep deprivation has

an effect on the immune system. Spiegel et al (2002) compared the number of antibodies produced by the body in response to the influenza vaccine in eleven healthy young men (allowed four hours sleep for six nights) and fourteen controls. The vaccination was given after the fourth night, and blood samples were taken immediately, and at ten and twenty-one days. After ten days, the sleep deprived men had significantly less antibodies than the controls in their blood. There was no significant difference at twenty-one days.

Energy conservation

Sleep can be an effective way to reduce energy consumption because the animal is immobile. Energy consumption can be measured by total metabolic rate or brain metabolic rate. This is the speed at which food is used by the body or brain as energy.

Ramanathan et al (2002) noted that high metabolic rate results in the generation of high levels of reactive oxygen species (ROS) which is linked to ageing. The researchers argued that longer periods of sleep are needed to interrupt the ROS process (and repair any damage to cells) in the brain. Smaller animals generally have higher metabolic rates, and the interruption of the ROS process could explain why they sleep longer.

FUNCTIONS OF REM SLEEP

REM sleep is often called "paradoxical sleep" because, though asleep, the brain and body are highly active. So energy conservation would not appear to be the reason for the evolution of REM sleep.

Learning and memory consolidation

As far back as the 1920s (Jenkins and Dallenbach 1924) was the idea established that sleep aids the consolidation of memories. This became specifically REM sleep in the 1970s ("REM consolidation hypothesis"). Research interest waned in the 1980s, and then was revived by animals studies using modern technology (eg Wilson and McNaughton 1994: hippocampal cells in rats in SWS). Winson (1993) proposed that theta waves in the hippocampus were key to encoding survival-enhancing information during waking and REM sleep.

REM deprivation studies are one of the main methods used here, and these are discussed later.

However, it should be noted that some mammals do learn despite limited amounts of sleep, and this is more likely that a "species uses sleep for learning if it can

afford to do so" (Hobson 2005).

Accepting that sleep deprivation reduces concentration and learning, Siegel (2005) was not convinced that sleep is crucial in memory consolidation.

One type of study involves enriched environments or learning before sleep which should lead to more REM sleep if the "REM consolidation hypothesis" holds. Animals studies, which are open to criticism, do show increased amounts of REM sleep, but not human studies (Vertes and Eastman 2000).

Development of the brain

The amount of REM sleep in a species negatively correlates with the level of maturity at birth. Mammals that are immature at birth (altricial) have much more REM sleep, both after birth and throughout the lifespan, than mammals mature at birth (pre-cocial) (table 4).

MATURE AT BIRTH	IMMATURE AT BIRTH
Guinea pig	Platypus
- born with teeth, claws and fur; moves within one hour and eats solid food by 1 day old	- newborn cannot move or regulate body temperature, and lives attached to mother
Adult - 1 hr of REM sleep (of 9 hrs total sleep)	Adult - 8 hrs of REM sleep (of 14 hrs total sleep)

Table 4 - Mature and immature at birth.

Long-term REM sleep deprivation studies from birth with kittens kept with one eye closed (Shaffery et al 1999), for example, produced less cells in certain parts of the brain. Thus, it seems, REM sleep is involved in the normal development of the brain.

Reversal of the effects of NREM sleep

This idea was first proposed as the "sentinel hypothesis" (Snyder 1966).

Periods of REM sleep follow those of NREM sleep, and their purpose is to reverse the negative effects of NREM sleep (Vertes 1986). During NREM sleep, activities of the body are "turned down" (compared to waking), and when woken from NREM sleep, animals are not alert. But waking from REM sleep is associated with immediate alertness which has evolutionary advantages.

Not only that, but if the physiology of the body is "turned down" in NREM sleep, the activation during REM sleep turns it up again.

Most importantly, it is the brainstem that is activated during REM sleep, and this controls the basic survival functions. Too much suppression of this part of the brain during NREM sleep could produce death. Thus the evolution of REM sleep to avoid this possibility (Wehr 1992).

Support for these ideas comes from the observation that marine mammals do not appear to have REM sleep. Because they are permanently moving, they never "slow down" their body, and so there is no need to "speed it up" again with REM sleep.

However, it should be expected that animals that need high levels of alertness, because of the high risk of predators, will have more REM sleep as part of total sleep, or shorter cycles of types of sleep during the night. On the other hand, there is a loss of muscle tone during REM sleep which paralyses an animal and increases vulnerability. "In general, mammals living in relatively exposed environments had short sleep times and a low density of REM sleep in both absolute and relative measures" (Lima et al 2005 p728).

SLEEP AND LEARNING AND MEMORY: RECENT RESEARCH

The benefits of sleep for memory and learning has only recently become a topic of research interest again since Karni et al (1994). This has become known as "sleep-dependent memory enhancement" (Stickgold 2005). The simple finding was that participants' recall and learning after a night of sleep is better than after the equivalent time awake. However, this is not without an opposing view (eg: Siegel 2005).

Research has shown that the relationship between sleep and memory is complex, and depends on what is learnt, and the stages of sleep. The studying of sleep and learning and memory makes use of a number of different methods and techniques:

i) Learning experiments - Participants learn a task and then they are retested later after sleep or not;

ii) Molecular studies - The increasing knowledge of genes, particularly from animal studies, has shown that certain genes

are active in the hippocampus at night. The hippocampus

is a key area of the brain related to learning and memory;

iii) Deprivation studies - Studies, again, on animals, deprived of light, for example, showed that cells in the brain changed during sleep (eg: Frank et al 2001);

iv) Neurophysiological studies - Measurements of brain activity (eg: EEG) showed that cells used in learning were active during sleep (eg: song learning in Zebra finch; Dave and Margoliash 2000);

v) Brain imaging studies;

vi) Dream studies - Subjective reports of dreams suggested that dreams are not just replaying episodic memories of the day, but are involved in learning; eg: Stickgold et al (2000c) analysed the dreams of participants after playing the computer game "Tetris".

Procedural Learning

This is the type of learning and memory used in perceptual and motor skills (like driving a car). Generally it improves with practice. It is studied by different tasks in experiments.

1. Visual texture discrimination

This task requires participants to identify diagonal lines among horizontal lines on the computer screen. It is a foreground-background discrimination task. It is the speed of identification that is measured, and this will improve with time.

Performance improves even more with sleep between training and testing compared to waking, but particularly the amount of slow wave sleep in the first quarter of the night (SWS1) and the amount of REM sleep in the last quarter (REM4) (Stickgold et al 2000a).

Calculations can be made by multiplying the percentage of these two types of sleep together. For example, when SWS1 x REM4 = 100, improvements in task time was an average of 15 milliseconds compared to 30ms where SWS1 x REM4 = 200 (Stickgold 2005).

Conversely, sleep deprivation the night after training stops improvements in performance, and this effect will continue after subsequent normal sleep (Stickgold et al 2000b). For example, the general improvement after three nights of normal sleep after training was around 20 ms, but only an improvement of

about five ms for participants who had one night of sleep deprivation followed by two nights of normal sleep (Stickgold 2005).

The improvement in performance due to sleep does not stop after one night, but continues for 48-96 hours afterwards if normal sleep occurs. For example, after the first night of sleep, there is an improvement of about 10ms, another 5 ms after the second night of sleep, and the same for the third and fourth night (and then the benefits peak) (Stickgold 2005).

2. Motor sequence task

This type of task includes asking participants to perform a particular sequence on a computer keyboard (eg: 7-9-A-Q-1), and their speed and accuracy are measured.

Walker et al (2002) measured the number of times a sequence could be achieved in thirty seconds 4-12 hours after training the same day, and after a night of sleep. The former saw an average increase of 4% from the baseline, and the latter a 20% increase. Also a ninety-minute daytime nap after training produced a 16% improvement in performance (Walker and Stickgold quoted in Stickgold 2005). Again improvements continued after the second and third nights of sleep, and were linked to a particular stage of sleep - in this case, stage 2 NREM in the last quarter of the night (Stickgold 2005).

Stickgold et al (2000b) found that sleep also reduced the number of errors (ie: increased accuracy) by 36% on these types of tasks compared to an increase of 9% for errors after twelve hours of daytime waking.

In a variation, Manoach et al (2004) compared chronic, medicated schizophrenic patients (n = 20) with controls (n = 14). The latter showed an overnight improvement on the task of 15%, but the former no improvement.

3. Motor adaptation task

This type of task involves activities like moving a cursor on a screen to draw a line, but the cursor moves independently of the controls. The participants must adapt to the unusual behaviour of the cursor. The number of errors are usually measured, and these are reduced by sleep after training. Improvements in performance are linked to slow wave sleep (Ghilardi et al 2000).

Tonini et al (2004; reported in Gorman 2005) interpreted the results from their similar experiment in a particular way. They argued that SWS weakens

connections between neurons rather than strengthens them as suggested by others. The reason is that the brain demands a lot of energy to maintain the connections between neurons. More learning leads to more connections which demands more energy, and soon the individual cannot supply enough energy per day (ie: eat enough food). During SWS, weaker connections are removed. This explanation is hypothetical at this stage.

Terry Sejnowski used this analogy: "When you fall asleep, it's like you're leaving your house and the workmen come in to renovate" (Gorman 2005 p47).

4. Serial reaction-time task

Participants are instructed to press keys on a computer in a particular sequence, which has a complex order that the participants are not told about. PET scans of sleeping individuals found that during REM sleep, regions of the brain active during learning of the task were also active (Maquet et al 2000). Sometimes the participants are warned that there is a pattern to the sequence of keys. They showed improvements in performance after sleep. But, interestingly, participants who did not know about the pattern showed improvements during waking as well as after sleep (Robertson et al 2004).

5. Auditory tasks

Results are slightly different here. For example, learning to recognise certain language sounds against the background of white noise showed improvements in accuracy after twelve hours either awake or asleep (Roth et al 2005).

In summary, procedural learning of perceptual and motor skills as shown by these tasks is improved by periods of sleep after training. Table 5 gives the main findings from these "sleep-dependent memory enhancement" studies.

FINDINGS
1. Performance improves after training with sleep compared to being awake.
2. These improvements continue with the second and third nights of sleep.
3. Different types of sleep are involved in different tasks.
4. Sleep deprivation immediately after training reduces performance even after two nights of normal sleep.
5. Daytime naps after training can improve performance.

Table 5 - "Sleep-dependent memory enhancement" studies and findings for procedural learning.

Complex Cognitive Procedural Learning

This is the learning of problem-solving techniques that tends to be relatively unique to humans (eg: insight learning). Wagner et al (2004) set participants mathematical problems to solve and gave them a technique for doing it. But there was a simpler technique, and the task was to see who would realise that. The participants were retested twelve hours after training. Of those who had remained awake during that period, around one-fifth realised the simpler technique compared to about two-thirds who had slept.

Declarative Memory

This is the memory for facts (semantic memory) and events (episodic memory). The tasks used to study this type of learning and memory are usually related to recall of words (eg: word-pairs).

The benefits of sleep are not so clear-cut here for memory improvement. A few studies showed early night slow wave sleep may enhance recall (Stickgold 2005), but other studies are not convinced (Smith 2001).

There are some researchers, however, who argued that REM sleep in particular improves recall of episodic and semantic memories. For example, Fiss et al (1977) found that recall of details of pre-sleep stories was improved if the participants had dreamt about the stories.

There are other ideas here including:

a) Stickgold et al (2001) REM sleep selectively strengthens associations of semantic memory;

b) Johnson (2005) dreaming produces "context memories" which are combinations of episodic memories, and this enables retrieval of episodic memories during waking;

c) Payne and Nadel (2004) the role of cortisol (stress hormone) released late in sleep, and strengthening of memories.

Spatial Memory

Spatial memory, using a virtual navigation task, does show overnight improvements (Peigneux et al 2004). Also studies with rats showed that hippocampus neurons used to learn the spatial task were active during REM sleep (Louie and Wilson 2001). This would support the function of REM sleep for memory consolidation. But, for

humans, it is during slow wave sleep that this neuronal process happens (Stickgold 2005). It seems that REM sleep for humans is linked to emotional memories (Wagner et al 2001).

Sleep Deprivation Studies

REM DEPRIVATION STUDIES

This type of study has been used to test the "REM consolidation hypothesis" (ie: REM sleep aids memory consolidation). Both animals and humans have been studied, and post and prior REM deprivation. Post studies involves REM deprivation after learning, and with prior studies, REM deprivation occurs before the learning. REM deprivation is relatively easy because the sleeper is woken only when their EEG shows the unique pattern of REM sleep, but they can have NREM sleep.

Animal Studies

i) Post REM deprivation studies

Animals are taught a behaviour, then deprived of REM sleep, and retested for recall. Studies tend to be equally divided in finding that memory is and is not disrupted by REM deprivation (Vertes and Eastman 2000).

The main concern of these types of studies related to the means of keeping the animals awake. They are placed on a platform in a water tank, and when in REM sleep fall into the water to wake them. The stress, for example, of this procedure is a confounding variable as well as the loss of all sleep (not just REM) with this technique.

b) Prior REM deprivation studies

Vertes and Eastman (2000) argued that studies depriving animals of REM sleep before training leads to performance, not necessarily memory, failures. In other words, the animals cannot physically perform the behaviour rather than having forgotten it.

Again the stress of REM deprivation techniques play as important a part as sleep loss itself. For example, van Hulzen and Coenen (1982) used two different techniques to deprive rats of REM sleep for three days. One technique was stressful (a platform in water), the other less so (a platform slightly off the ground, and the rats fall off when in REM sleep). Recall varied depending on the technique of deprivation used - it was

worse for the more stressful technique.

Horne (1988) found both types of animals studies here "unconvincing". But Smith (1996) has proposed "REM windows". These are specific parts of REM sleep that are involved in memory consolidation. There is a dispute about this idea, especially for humans (Vertes and Eastman 2000).

Human Studies

Post and prior REM deprivation studies have also been performed on humans. But different types of studies have been used as well.

i) Effects of anti-depressant drugs

Anti-depressants tend to suppress REM sleep. If the "REM consolidation hypothesis" is correct, these drugs should also reduce recall. For example, one type of anti-depressant, monoamine oxidase inhibitors (MAOI), stopped REM sleep for months (eg: 226 days in one patient; Wyatt et al 1971). But memory was not affected.

Vertes and Eastman (2000) reviewed studies on all types of anti-depressants, and found that, despite reduced REM sleep, memory and cognitive functioning were not disrupted.

ii) Brain injured patients

There are a few cases of individuals with brain injury that stops REM sleep, but does not cause other major problems (eg: Osorio and Daroff 1980 - bilateral pontine lesions). These individuals seem to be able to live normal lives.

Lavie et al (1984) reported a case of a 20-year-old man who had gunshot damage to the pons. Despite being in a critical condition after the shooting, he recovered enough to qualify and work as a lawyer.

Solms (1997) studied patients with "global anoneria" (total loss of dreams) or "visual anoneria" (partial loss of dreams), both caused by injuries to different areas of the brain.

REAL-LIFE STUDIES

Opportunist studies have been made of individuals voluntarily staying awake for very long periods, as in world record attempts: well-known examples like Peter Tripp (Luce and Segal 1966) and Randy Gardner (Dement

1978), or the "rocking chair marathon" of 449 hours (18 days 17 hours) by Maureen Weston in 1977 (Pinel 2002).

Peter Tripp

In 1959, in New York City, WMGM radio DJ, Peter Tripp attempted to stay awake for 200 hours (8 days 8 hours) ("wakethon") to benefit a charity. During this time, he was broadcasting his daily show from a glass-walled booth in Times Square.

Because of the concern for his health, Tripp was well studied throughout the 200 hours by doctors and psychiatrists (Floyd Cornelison and Louis West). There was considerable interest from sleep researchers as the knowledge about sleep (and sleep deprivation) was limited at that time. The knowledge varied from animals studies in the 1890s, which showed that dogs died after 6-13 days without sleep, through to US military claims that their soldiers could stay awake for ten days with no adverse effects (Coren 1996).

After two days, Tripp stated hallucinating; eg: "Specks on the table began to look like bugs. He thought he saw a rabbit in the booth" (Luce and Segal 1966 p91). Some of the hallucinations were linked to paranoia, like a hotel desk was on fire (after 120 hours).

On the last day, he mistook a doctor (Dr.Wolff) with an umbrella as a funeral director come to measure him for a coffin.

Memory and concentration problems increased with time - by 170 hours, he could not say the whole alphabet. By 150 hours awake, he was disoriented. Yet he still managed each day to do his three-hour radio show (5-8pm) effectively. In fact, some listeners had no idea he was not sleeping.

At the end of the 200 hours (201 exactly; Coren 1996), he slept for thirteen hours, and awoke apparently refreshed, though he did have mild depression for three months (Luce and Segal 1966).

Tripp believed that there were no long-term consequences, but he lost his job and his marriage soon after ("Altered States" 1998).

Randy Gardner

In January 1964, 17 year-old student, Randy Gardner, attempted to stay awake for 264 hours (11 days) as a project for the San Diego Science Fair.

Some textbooks reported that Randy had few problems from the sleep deprivation, but Coren (1996) felt that was inaccurate. Daily examinations were made by

Lt.Commander J.J.Ross of the US Navy Medical Neuropsychiatric Research Unit. Here are some of the main observations of problems that came and went (quoted in Coren 1996):

Day 2: Visual problems which made watching television impossible;

Day 3: Mood changes, and physical co-ordination problems;

Day 4: High irritability; memory and concentration problems; hallucinations (eg: street sign was a person) and delusions (eg: believed he was famous US football player);

Day 5: Hallucinations less disturbing as he was now aware what they were; general improvement;

Day 6: Relapse to day 4;

Day 7: Slurred speech, and reappearance of high irritability;

Day 8: Memory and concentration problems most apparent;

Day 9: Fragmented thinking (eg: unable to finish sentences);

Day 10: Paranoia - eg: thought radio DJ who interviewed him was against him;

Day 11: Physical co-ordination reasonable; slurred speech; memory problems.

Afterwards Randy slept for 14 and 3/4 hours, and awoke reasonably refreshed. On the second night, he slept for twelve hours, and 10 hours on the third night. His normal length of sleep (8 hours) returned by the next night. He mostly recovered stage 4 NREM sleep then REM sleep (Horne 1988).

LAB STUDIES

Lab studies allow more controlled observation of sleep deprived individuals than real-life studies, but usually for shorter periods because of ethical concerns for the participants.

Drummond et al (2000) is a good example of a recent study which makes full use of modern technology in the lab. Thirteen young adults were given functional magnetic resonance imaging (fMRI) scans after thirty-five hours of sleep deprivation. Different parts of the brain were active during memory tests compared to after sleep. The prefrontal cortex and areas of the parietal lobe were more active, and areas of the temporal lobe less active after sleep deprivation. This was interpreted as compensation by the brain. However, average recall of nouns dropped from 4.7 words to 2.8 after sleep deprivation.

Mechanisms of Sleep

FALLING ASLEEP

Cells in the basal forebrain, brainstem, and hypothalamus are all involved in the daily cycles of sleep and wakefulness (Saper et al 2005). This is seen in the idea of an "arousal pathway" in the brainstem (from the pons to the midbrain).

This was first noted by Von Economo (1930), in Vienna, when he came across patients with a form of "sleeping sickness" (encephalitis lethargica). Sufferers slept for over twenty hours per day. Post-mortem studies of the brains of sufferers showed damage at the junction of the midbrain and the diencephalon.

More recent work has identified two branches of the "arousal pathway" (Saper et al 2005):

i) Upper brainstem to thalamus (pedunculo-pontine and laterodorsal tegmental nucleus; PPT/LDT): cells active during wakefulness and REM sleep;

ii) Upper brainstem to lateral hypothalamus and basal forebrain (bypassing thalamus): lesions produced sleepiness and even coma. Neurons tend to fire most during wakefulness, less in NREM sleep, and stop during REM sleep.

Von Economo also found patients with the "sleeping sickness" who slept very little despite being tired. These patients had damage to the basal ganglia and anterior hypothalamus. Later animal research (eg: Lu et al 2000) found that damage specifically to the ventrolateral preoptic nucleus (VLPO) reduced sleep by 50%.

It is now felt that these areas in the brain are part of a "flip-flop (sleep) switch" (Saper et al 2001). This is a switch that in the "on" position produces wakefulness, and in the "off" position, sleep.

The switch will change relatively quickly, which has evolutionary advantages - ie: half-awake states with low levels of alertness are risky for animals. The inappropriate flipping of the switch can be linked to narcolepsy.

DIFFERENT TYPES OF SLEEP

NREM sleep is linked to areas in the forebrain (eg: hypothalamus) through the presence of "sleep-active"

neurons. Stimulating or heating these neurons induces sleep, while damage to them reduces sleep (Siegel 2005).

Neuroimaging studies show a general decrease in brain activity in NREM sleep (varying from 11-40% in glucose utilization) compared to REM sleep and waking. This is centred on the brainstem and thalamus (Hobson et al 2000).

REM Sleep

REM sleep has specific neurons in the brainstem: "REM sleep on" neurons in the pons, and "REM sleep off" neurons in the pons and medulla. "REM sleep off" cells are continuously active during waking, reduced activity during NREM sleep, and silent during REM sleep.

The "REM sleep on" cells produce a loss of muscle tone (ie: paralysis) (Siegel 2005). Injecting them with a particular neurotransmitter (acetylcholine agonist) induces prolonged REM sleep in mice, for example (Coleman et al 2004) as does cooling them in cats (Jouvet et al 1988 quoted in Siegel 2005). While damage to the cells reduces or stops REM sleep.

Generally cells in the brain (eg: brainstem) show as much activity during waking as during REM sleep.

Also during REM sleep, activity in the hippocampus is increased relative to waking and NREM sleep (Nielsen and Stenstrom 2005).

The development of neuroimaging has allowed researchers to "see inside" the sleeping brain. During REM sleep, the following areas are active: pons, amygdala, and hypothalamus, while parts of the visual and frontal cortex are inactive (Vertes and Eastman 2000). Braun et al (1998) has called this a "closed system" disconnected from external inputs and outputs (ie: outside the brain).

Though a lot can be learnt from neuroimaging studies, Hobson et al (2000) pointed out that there are limitations like the measurement of brain activation used (eg: glucose or oxygen uptake). Furthermore, sleep deprivation associated with many sleep studies could change the physiology of the brain.

Dreaming

The assumption since the 1950s is that REM sleep is dream sleep. In fact, it was initially called "D-sleep" (Dement and Kleitman 1957). Though it is also known that individuals woken from NREM sleep report dreams in a small number of cases (eg: 5-10%; Hobson 1988). Reports from REM sleep awakenings are longer (eg: seven times

longer; Stickgold et al 1994).

Recently, Solms (2000) has argued that REM sleep and dreaming were linked but were separate states that could occur independently. He proposed two pieces of evidence:

i) A larger number of dreams occur outside REM sleep. Solms felt "roughly one-quarter", particularly in early NREM sleep (stage 1) and later NREM sleep before waking. Nielsen (1999), in a review of studies, found that on average 81.8% of individuals woken from REM sleep recall dreams compared to 42.5% in NREM sleep.

ii) Different areas of the brain control REM sleep and dreaming. This can be shown by studies of brain injured patients. Damage to areas of the brain known to control REM sleep does not stop dreaming, and damage to other areas of the brain stops dreaming.

For example, nine patients with damage to the cortical-limbic circuit in the forebrain had REM sleep but no dreams (reported in Hawkes 1999).

Solms argued that dreaming originated in the forebrain including the hypothalamic and amygdaloid areas, and parts of the cortex. Dreaming can also be affected by manipulation of the neurotransmitter dopamine, and cause no change to REM sleep.

MOTIVATION TO SLEEP

The motivation to sleep seems to be based upon two drives - homeostatic and circadian (Borbely and Achermann 2000):

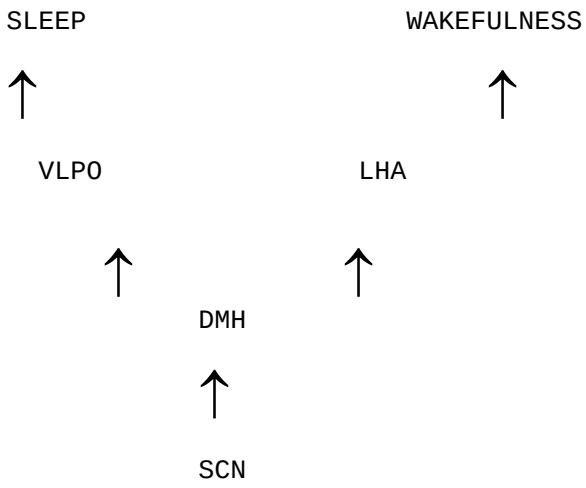
i) Homeostatic drive (process S)

This is based on the idea of balance or need. In other words, a certain amount of sleep is needed. During waking, there is a build-up of the "need to sleep", and this is dissipated with sleep.

As to what this build-up substance could be, it is not clear (Saper et al 2005). In the past, for example, factor S was proposed, but, most recently, it is adenosine. For example, injection of adenosine into the basal forebrain of cats caused sleep (Strecker et al 2000).

ii) Circadian regulation (process C)

This sees sleep as due to the 24-hour circadian cycle, which is controlled by the suprachiasmatic nucleus (SCN) (Reppert and Weaver 2002). The process of control by the SCN is complex, figure 1 gives the simplified version.



SCN = suprachiasmatic nucleus
DMH = dorsomedial nucleus of hypothalamus
LHA = lateral hypothalmus
VLPO = ventrolateral preoptic nucleus

(After Saper et al 2005)

Figure 1 - SCN and other brain areas in circadian regulation of sleep and wakefulness.

Both these processes in motivating sleep can be altered by external cues like availability of food or emergency, or overcome by choice in humans. McEwen and Stellar (1993) proposed an "allostatic drive" for sleep to explain the changing amounts and timing of sleep when external events demanded it. In other words, the arousal system can override the homeostatic and circadian drives to produce "hyperarousal" in emergencies.

But this may happen in unwanted situations, like insomnia. Nofzinger et al (2004) found increased activity in parts of the cortex of sleeping humans with insomnia compared to non-insomniacs (using PET scans).

Sleeping Disorders

Studies of sleep disorder sufferers, as well as helping these individuals, can also throw light on sleep generally. Mahowald and Schenck (2005) noted two such examples: "sleep may not be a global, but rather a local, brain phenomenon", and that wakefulness, REM and NREM sleep may not be mutually exclusive states. This latter situation can be seen in "ambiguous sleep" where EEG readings show both REM and NREM sleep simultaneously.

The number of sleep disorders (or more correctly, sleep/wake disorders) is nearly one hundred. But for convenience, they are categorised as hypersomnia, insomnia, circadian rhythm disorders, and parasomnias (Mahowald and Schenck 2005).

HYPERSOMNIA

This refers to disorders involving excessive daytime sleepiness; in particular caused by obstructive sleep apnea (associated with snoring), and narcolepsy. It does not include "volitional sleep deprivation"; ie: the choice to have less or no sleep.

Narcolepsy

This involves sudden sleepiness, cataplexy (muscle weakness), hallucinations (either at the onset of sleep - hypnagogic - or on waking - hypnopompic) (1), and sleep paralysis (feeling awake but paralysed when still asleep).

Sufferers do not sleep overall more than the average, but "they are unable to keep the normal boundaries of wakefulness, NREM sleep and REM sleep" (Mahowald and Schenck 2005). For example, automatic behaviour (eg: unwrapping a sweet, throwing the sweet away and putting the wrapper in the mouth) is a mixture of wakefulness and NREM sleep. While sleep paralysis and cataplexy are the combination of REM sleep and wakefulness.

Narcolepsy is relatively rare affecting one in two thousand people (Mahowald and Schenck 2005). Though rates vary between 1 in 600 in Japan to 1 in 500 000 in Israel (Siegel 2000). It seems to be related to a gene on chromosome six (Mignot 1998) for humans. While animal studies have found a link to the "sleep switch".

The flipping of the "sleep switch" inappropriately could cause this condition. Recent research has found

that this switch is controlled by neurocehmicals called orexins or hypocretins (eg: Lin et al 1999). Orexin is only made in the hypothalamus (Siegel 2000). Human sufferers of narcolepsy (with cataplexy) were found to have less orexin neurons (based on post-mortems) (eg: Thannickal et al 2000). Orexin appears to encourage arousal.

One suggestion is that orexin neurons are reduced because the immune systems of narcoleptics are attacking them mistaken as foreign substances (autoimmune disease) (Siegel 2000).

INSOMNIA

These conditions relate to problems in falling asleep and/or staying asleep. Insomnia can be caused by many things, from depression to caffeine, but work has focused on hyperarousal. Comparisons of chronic insomniacs and non-insomniacs over twenty-four hours has shown differences - the former have less daytime sleepiness (Bonnet and Arand 2000). This is tested by leaving individuals in a quiet, dark room for short periods during the day to see if they fall asleep (known as the multiple sleep latency test; Zeman et al 2001). The hyperarousal has been linked to increased secretion of stress hormones (like cortisol and adrenocorticotrophic hormone) (Vgontzas et al 2001).

A rare type of insomnia is Fatal Familial Insomnia (FFI), which is a genetic condition leading to degeneration of the thalamus due to the prion protein. It is probably a form of transmissible spongiform encephalopathy (TSE) (eg: Creutzfeld-Jakob disease: CJD) (National Institute of Neurological Disease and Stroke 2005).

The malfunctioning of the thalamus stops sleep, disrupts the circadian rhythms, and produces dementia. The result of the disease is death. There are four stages to its progression lasting about eighteen months (Akroush 1996-7):

- Progressive insomnia including panic attacks and bizarre phobias (approximately four months);
- Continuing insomnia with hallucinations, panic, and agitation (five months);
- Total insomnia (three months);
- Dementia, total insomnia, and death (six months).

FFI does not show itself until later adulthood. In an Italian family, where twenty-nine of 288 relatives were affected, the average age of onset was forty-nine (Akroush 1996-7). Because FFI is a rare genetic mutation, it tends to remain in families as in the Italian case,

and a Chinese family (Spacey et al 2004).

One problem with insomnia is that individuals report less sleep than they actually had. This is because individuals notice being awake more.

Semler and Harvey (2005) led insomniac students to believe that they had a bad night's sleep when they had not. These students showed behaviour as if they had not slept well (eg: negative thoughts, feeling sleepy). The researchers argued that it is the anxiety about not sleeping well that can be as important as actually not sleeping well for some insomniacs.

One cause of severe insomnia is restless legs syndrome. This involves unpleasant sensations in the legs when inactive, particularly during the period of falling asleep. The sensations are reduced if the individual moves their legs, including walking about. It affects between 5-15% of the population (Phillips et al 2000).

Explanations for restless legs syndrome include genetic (Desautels et al 2005), iron deficiency (Allen and Earley 2001), or reduced dopamine in part of the brain (Turjanski et al 1999).

CIRCADIAN RHYTHM DISORDERS

Here sleep problems are related to the body's biological clock (also known as wake-sleep schedule disorders). This is manifest as sleeping at the wrong time or the inability to sleep at the right time.

Primary wake-sleep schedule disorders are due to problems with the biological clock (in the brain), while secondary disorders are a product of environmental change (eg: jet-lag). Examples of circadian rhythm disorders include delayed sleep-phase syndrome (DSPS) (sleeping and waking late), and advanced sleep-phase syndrome (ASPS) (sleeping and waking early).

Xu et al (2005) constructed a family tree for ASPS to show the genetic basis (PER 2 gene). The sufferers fell asleep early in the evening (eg: 7-8pm) and woke early the next morning (eg: 4-5am). Onset of the condition occurred between early childhood and the mid-teens. In the family, the grandmother was a sufferer, and so were three of her four daughters, and one granddaughter.

PARASOMNIAS

This category relates to behaviour occurring during sleep. For example, wakefulness and NREM sleep

simultaneously as in sleepwalking or sleep terrors, and wakefulness and REM sleep together in REM sleep behaviour disorder (RBD).

NREM Sleep

These parasomnias tend to occur in stages 3 and 4 of NREM sleep (also known as slow-wave sleep). More common ones, like sleepwalking, through to rare examples like sleep-related eating (Winkelman et al 1999) (table 6).

CONDITION	PREVALENCE	STUDY
Confusional arousal	4% adults	Ohayon et al (1999)
Sleepwalking	1-17% children 4% adults	Klackenberg (1982) Ohayon et al (1999)
Sleep terrors	3% adults	Ohagon et al (1999)

Table 6 - Prevalence rates of NREM parasomnias.

REM Sleep

The most common example here is REM sleep behaviour disorder (RBD), where individuals act out their dreams (often violently). The majority of sufferers are male (90%), and over the age of fifty years (Mahowald and Schenck 2005).

One suggestion is that RBD is an early manifestation (by ten years) of neurodegenerative conditions, like Parkinson's disease (Boeve et al 2003).

Dreams

Nielsen and Stenstrom (2005) described dreams as "an unexplained marvel of human existence". Freud's (eg: 1949) theory of dreams has dominated any theory to explain the meaning of the dream contents (2).

There are many and varied theories of the meaning of the content of dreams. But, in practice, the theories either see the dream as meaningful or not. Table 7 gives some examples of theories of dreams.

DREAMS ARE MEANINGFUL	DREAMS ARE MEANINGLESS
- Historical/cultural beliefs eg: Toraja (Indonesia) naked in dream means will get sick; receive gold means good rice harvest (Weiten 1995)	- Activation-synthesis hypothesis (Hobson and McCarley 1977; Hobson et al 1998)
- Psychoanalysis: real meaning hidden	- Cognitive view (Evans 1984)
- Message to self: eg: brain picks up signals from immune system about impending illness before conscious awareness (Aldridge 1999)	PARTLY MEANINGFUL/PARTLY MEANINGLESS
- Beneficial to self eg: in learning or problem-solving (Stickgold 2005) or emotionally (eg: Cartwright et al 1998; Kramer 1993)	- "Mental Housecleaning" hypothesis (Crick and Mitchinson 1983)
- Predicting future (eg: Ullman et al 1989)	

Table 7 - Different types of theories about dream content.

Methods used to study dreams

i) Pre-sleep stimulation - This type of experiment involves stimulating the participants in a particular way before sleep; for example with a provocative film or computer game, and then seeing how much of the pre-sleep event is reported in dreams.

Interestingly, individuals in a sleep lab for the first time, not only have poor sleep, but report dreams with content related to the lab environment ("first night effect") (Hobson et al 2000).

ii) Sensory stimulation during sleep - In this form of experiment, the sleeper during REM sleep is stimulated in some way (eg: noise, smell) that does not wake them, to see if that stimulation appears in the dream content.

iii) Animal studies - Louie and Wilson (2001) found that animals taught particular behaviour (like maze learning) before sleep showed a replay of that behaviour as measured by the firing of neurons in the hippocampus in real-time during REM sleep.

iv) Brain-injured patients - Torda (1969) reported the cases of three patients with damage to the hippocampus. Their waking episodic memory was poor. The individuals rarely reported dreams, but, if they did, the dreams were short, repetitive and unemotional. In some cases, the dream was a replay of an actual event, whereas normal dreaming tends to be more of an elaboration of the events. However, it has to be asked whether the memory problems affected the recall of dreams to the researcher.

Dreams and events of the day

The current question relating to dreams is whether their content mirrors the day's experiences (in some way). Recent research has focused upon the relationship between dream content and the previous day's events (as recalled in episodic memories).

Based on analysis of dream recall and linking it to events of the day, studies produced varying figures - from 1.4% to 65% (Fosse et al 2003).

Not all dreams are related to the previous day, but they can be older memories. Nielsen et al (2004) found qualitative differences in dreams based on the previous day's events and older ones. Participants who watched a distressing film about the ceremonial sacrifice of a water buffalo produced a U-shaped curve in terms of dream content.

Many details of the film in the first three nights of sleep afterwards, then a reduction on the fourth night, and an increase in content on the fifth to seventh nights.

"This is consistent with the notion that dreams draw upon memories at different stages of consolidation and from different regions of the brain" (Nielsen and Stenstrom 2005 p1288).

Studies with animals showed that newly-formed memories occur in the hippocampus, and take about a week to relocate to other parts of the cortex (eg: Thompson et al 1996).

Dreams are not just about recalling events from waking, but have an emotional content. Emotions generally are linked to the amygdala in the brain, and this is more active during REM sleep then during waking (Hobson et al 1998). This would explain the emotional content of dreams, particularly in nightmares.

Other researchers have moved away from dreams as accurately representing waking events. Fosse et al (2001) asked participants to report their mental experiences during five different periods of the 24-hour day: active wakefulness, quiet wakefulness, sleep onset, woken from NREM sleep, and woken from REM sleep. The reports were scored on a scale of hallucinatory perception or thinking. A negative correlation was found between the two. During wakefulness hallucinatory perception was lowest and thinking highest, and the opposite for REM sleep. The authors took this as evidence of dreaming as hallucinatory process.

Hobson (2005) argued further that "REM sleep is a physiological brain state that produces a distinctive and psychosis-like mental content, whereas during normal waking such properties are suppressed" (p1256). It is not clear why this is so. Hobson supported this idea with an unpublished experiment he was involved in (Scarone et al). They found that schizophrenic participants achieved high scores on a bizarreness scale when reporting thoughts awake and woken from REM sleep, while non-schizophrenic participants only gained a high score when reporting their dreams.

Footnotes

1. Ohayon et al (1996) telephone surveyed 4972 individuals aged fifteen years and over in the UK, and 37% reported experiencing hypnagogic hallucinations and 12.5% hypnopompic hallucinations, at least twice weekly. Both types of hallucinations were related to sleep problems, like insomnia (table 8).

	Hypnagogic (%) Hallucinations*	Hypnopompic (%) Hallucinations*
Difficulty falling asleep	49.5	16.7
Difficulty staying asleep	49.7	17.2
Waking early	51.3	19.5
Nightmares (more than one per month)	65.1	21.8
Difficulty waking at right time	54.9	20.3

(* All significant differences at p = 0.05 compared to alternatives)

(After Ohayon et al 1996)

Table 8 - Sleep difficulties and hallucinations.

2. Solms (1997) has updated Freud's ideas to include modern neuropsychological knowledge from brain injured patients.

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